UNITED STATES DEPARTMENT OF COMMERCE
Patent and Trademark Office
Address: COMMISSIONER OF PATENTS AND TRADEMARKS
Washington, D.C. 20231

MU

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.
08/670,119	06/25/96	NG	G SIM-001(7434

HM22/1129

PATENT ADMINISTRATOR
TESTA HURWITZ & THIBEAULT
HIGH STREET TOWER
125 HIGH STREET
BOSTON MA 02110

EXAMINER	
HAYES,R	
ART UNIT	**PAPER NUMBER**
1647	25

DATE MAILED: 11/29/00

Please find below and/or attached an Office communication concerning this application or proceeding.

Commissioner of Patents and Trademarks

UNITED STATES DEPARTMENT OF COMMERCE
Patent and Trademark Office
ASSISTANT SECRETARY AND COMMISSIONER OF
PATENTS AND TRADEMARKS
Washington, D.C. 20231

BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

Paper No. 25

Application Number: 08/670119
Filing Date: June 25, 1996
Appellant(s): Ng et al.

Michael Twomey
For Appellant

EXAMINER'S ANSWER

This is in response to appellant's brief on appeal filed August 28, 2000.

(1) Real Party in Interest

A statement identifying the real party in interest is contained in the brief.

(2) Related Appeals and Interferences

A statement identifying the related appeals and interferences which will directly affect or be directly affected by or have a bearing on the decision in the pending appeal is contained in the brief.

(3) Status of Claims

The statement of the status of the claims contained in the brief is correct.

(4) Status of Amendments After Final

The appellant's statement of the status of amendments after final rejection contained in the brief is correct.

(5) Summary of Invention

The summary of invention contained in the brief is correct.

(6) Issues

The appellant's statement of the issues in the brief is correct.

(7) Grouping of Claims

Appellant's brief includes a statement that claims 18, 20-37 and 60-65 do not stand or fall together and provides reasons as set forth in 37 CFR 1.192(c)(7) and (c)(8).

(8) Claims Appealed

The copy of the appealed claims contained in the Appendix to the brief is correct.

(9) Prior Art of Record

The following is a listing of the prior art of record relied upon in the rejection of claims under appeal.

5,508,384	MURPHY et al.	4-1996

Lofts et al. "Specific short transmembrane sequences can inhibit transformation by the mutant *neu* growth factor receptor *in vitro* and *in vivo*." Oncogene, vol. 8 (1993), pp 2813-2820.

Rudinger, J., In "Peptide Hormones" (ed. J.A. Parsons) University Park Press, Baltimore (June 1976), pp. 1-7.

THE MERCK MANUAL of Diagnosis and Therapy. (ed. Robert Berkow) Merck Research Laboratories, Rathway, NJ (1992), pp. 2657.

(10) Grounds of Rejection

The following ground(s) of rejection are applicable to the appealed claims:

1. Claims 18, 20-37 & 60-65 stand rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

The specification proposes specifically inhibiting transmembrane receptor function of D2 and D1 receptors, β1 and β2 and α1A adrenergic receptors, EGF-tyrosine kinase receptors, dopamine transporter proteins, GABA-specific ion channel receptors, a T-cell antigen receptor, as well as vasopressin, serotonin and angiotensin receptors, in mammalian disorders through administering transmembrane hydrophobic peptides that specifically interact with the transmembrane regions of these receptors. However, it is unknown, nor disclosed, what the metes and bounds of the recitation "treating... a disorder" in a mammal entails; nor how one would know when, or if, they have successfully practiced the invention "in a mammal", as broadly claimed, using any peptide, or any biologically functionally equivalent fragment or analogue of such, that "consists essentially of at least four consecutive amino acid residues from the amino acid sequence of at least one [structurally unknown] transmembrane domain of the [structurally unknown] integral membrane protein..."; especially when no disorder "is indicated".

The instant specification also provides no description nor guidance on what "intracellular" membrane receptors are envisioned to be specifically affected (e.g., within the endoplasmic

reticulum or in lysosomes, as mentioned on page 7; as it relates to claim 20), nor what specific peptides could work in this claimed embodiment. Nor is any description or guidance provided within the specification that distinguishes the metes and bounds of (c) an ion channel, versus (d) an ion channel receptor, such as the GABA-A receptor, versus (e) a channel protein (i.e., as it relates to claim 22). Nor is any description or guidance provided within the specification (e.g., on pages 20 and 22) that discloses what "substance abuses" are envisioned to be treatable (i.e., as it relates to claim 28).

Moreover, a method of treating any disorder that putatively involves altered integral membrane protein expression requires administration of specific peptides within sufficient proximity of the affected cell population to have any putative effect. In contrast, the specification provides no guidance on how to determine when a patient is in need of such treatment; nor what symptoms are envisioned to be treated; nor how the severity of these symptoms is related to the efficacy of an integral membrane receptor expression; nor how one would know when such administration is appropriate (e.g., especially as it relates to the disorders of claim 28 that have no known origin, nor art recognized "specific therapy"; see Merck Manual, page 2657 for Huntington's disease, for example). Therefore, the skilled artisan cannot successfully predict or reasonably determine when, or if, the instant invention works *in vivo*, because the parameters that need to be addressed for assaying whether the instant invention is "effective' in "treating, in a mammal, a disorder... for which administration of an antagonist... is indicated" are not disclosed, or not known, except for possibly decreasing heart rate using a β1-adrenergic-specific peptide

(see page 41), which is further not specifically claimed. Additionally, although an assay is described that induces asymmetric body posture in rats by acting as a D2 antagonist following intracerebral injection into the striatum, a disease state such as Parkinson's disease is characterized by dopamine receptor inactivity, versus over-activity, which contradicts the feasibility of the instant invention for "treating... disorders" generically, as "indicated". Second, in that vehicle gave a comparable change in blood pressure as the β1-adrenergic-specific peptide (page 42), treatment of hypertension does not appear to work using these transmembrane peptide molecules (i.e., as it relates to claims 33 and 35). In summary, no appropriate functional assays are provided for determining when treatment is effective, or when needed; especially for treating any uncharacterized disorder that may, or may not, involve dysfunctional expression of an integral membrane protein.

Alternatively, the claims are not enabled because no universal treatment is recognized within the art for "treating... disorders", in general, in that the mechanism resulting in a disorder by one causative factor is not predictive of the mechanism/treatment of a disorder by a different causative factor, which may not involve altered integral membrane receptor expression. For example, disorders of the nervous system include neuronal cell damage that often results in cell death (e.g., see Merck Manual, pg. 2657; as it relates to claim 28). Accordingly, there is no reasonable expectation from one of ordinary skill in the art that merely administrating transmembrane-specific peptides can "effectively" "treat" neurological conditions characterized by dead neurons (e.g., Huntington's disease; as it relates to claim 28). Thus, treatment of

schizophrenia, Huntington's disease, Tourette's syndrome and any general substance abuse, as encompassed by the current claims, is also not reasonable because effective treatment for such neurological disorders is not known in the art, nor adequately described within the instant specification, nor adequately claimed; thereby, requiring undue experimentation to discover what constitutes an "effective amount of an antagonist peptide" to be administered.

Lastly, the name "effective fragment or analogue thereof" (as it relates to how it is defined on pages 7-9 of the specification), does not sufficiently characterize and enable the peptides that are encompassed by the claims, because the inclusion of any "biologically functional equivalent" within the definition of "fragments or analogues" of a integral transmembrane peptide sets forth no structural characterization and little functional characteristics (i.e., as it relates to claims 18, 25-26, 30 & 33). In contrast, the specification does not teach which particular amino acids are critical for any integral transmembrane peptide's function, nor what structural features distinguishes the claimed peptides from any other peptide without the desired function of the instant invention. The specification also does not provide any evidence that any "fragment or analogue" of any transmembrane peptide possesses the desired biological activity of a receptor antagonist, except for possibly the two peptide fragments depicted in SEQ ID NOs. 30 and 31. Therefore, because the specification does not disclose those amino acid residues that are critical for inhibiting function of integral membrane receptors, nor which residues can be altered and still maintain the desired functional activity of the instant invention (i.e., as it also relates to conservative substitution variants thereof), the resultant random mutations and truncations to

peptides with limited characterization would be predicted by the skilled artisan to result in inactive peptides. For example, Rudinger states on page 3 that "it is impossible to attach a unique significance to any residue in a sequence. A given amino acid will not by any means have the same significance in different peptide sequences, or even in different positions of the same sequence". Rudinger further states on page 6 that "the significance of particular amino acid sequences for different aspects of biological activity cannot be predicted *a priori* but must be determined from case to case by painstaking experimental study". Thus, the lack of guidance provided in the specification, as to what minimal structural requirements are necessary for inhibiting function of integral membrane activity, or assays to determine such, would prevent the skilled artisan from determining whether any peptide "fragment or analogue" could be made that retains the desired function of the instant invention for treating "indicated" "disorders", because the 3-dimensional conformation (i.e. the helical conformation) of a native transmembrane protein would be predicted to be adversely altered without requiring undue experimentation to determine otherwise.

2. Claims 18, 20-37 & 60-65 stand rejected under 35 U.S.C. 112, second paragraph, as being indefinite and incomplete for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention. It remains unclear when "administration of an antagonist ... is *indicated*" when no such step is recited in the claims, and when it is unknown what "disorder" is to be "treated" when none is recited, or "indicated", in the claims.

Claims 18 and 36 (as well as all dependent claims) stand rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention for the recitation, " consisting essentially of... amino acid residues", because a peptide is defined by its amino acid sequence in which different sequences, such "conservative amino acid substitution variant[s]" change the peptide being claimed, by definition, and therefore, cannot also "consisting essentially of", by definition.

3. Claims 18, 20-22, 36 & 60-61 stand rejected under 35 U.S.C. 102(b) as being anticipated by Lofts et al. (IDS REF# AN).

Lofts et al. teach treatment of nude mice with an effective amount of encoded WT peptide sequences (see pages 2814, 2816-2817; Figures 1 & 6), which "consists essentially of at least four consecutive amino acid residues from the amino acid sequence of... at least one transmembrane domain" of the mammalian *neu*/EGF integral plasma membrane protein, such that growth of solid tumors in these mice was reduced (pages 2816-2817), in which the essential part of Lofts peptides is the presence of a transmembrane domain (i.e., pentapeptide residue #s 661-665; see Abstract; as it relates to the recitation, "consists essentially of").

4.. Claims 18, 20-24, 29, 36-37 & 60-61 stand rejected under 35 U.S.C. 102(e) as being anticipated by Murphy et al. (U.S. Patent No. 5,508,384).

Murphy et al. teach use of dopaminergic (col. 13, lines 29-49; i.e., as it relates to claims 22-24) and adrenergic (col. 16 line 60-col. 27, line 10; i.e., as it relates to claims 22-23 & 29) G-protein-coupled transmembrane receptor peptides (i.e., peptides from an integral membrane protein) in pharmaceutical compositions to "treat" G-protein-related diseases, such as schizophrenia (e.g., cols. 35-37; as it relates to claims 18, 20-21, 36-37 & 60-61), which is equivalent to the actions of an "antagonist", by definition, when "inhibition of binding of Dopamine D2 receptors" occurs (col. 36, lines 12-17). It is noted that column 36, lines 14-16, states "using a GPR polypeptide corresponding to a fragment or consensus portion of a dopamine D2 *transmembrane domain* [emphasis added]", and that column 35, lines 41-42, states that "peptides of the present invention are generally small (10-40... amino acids); thereby, meeting all essential and structural limitations of these claims (i.e., as it relates to "consisting essentially of at least four consecutive amino acid residues... from the amino acid sequence of at least one transmembrane domain"), which inherently and "specifically inhibits the activity of the integral membrane protein", as claimed; absent evidence to the contrary.

(11) Response to Argument

Issue 1: Appellants argue on pages 5-7 of the Brief that "[t]he ordinary skilled artisan will understand, having read the specification, that once he or she is faced with a disorder in which, for example, one wishes to inhibit the activity of a particular integral membrane protein of known amino acid sequence, one can then fashion a very specific antagonist peptide to control activity of

that integral membrane protein by following the teachings of the specification to select a suitable antagonist peptide", and that "[i]t is not necessary for the Applicants to teach every possible disorder in which these is a need for inhibition of an integral membrane protein". Appellants then argue on pages 6-7 that "[t]he specification teaches precisely what antagonists are expected to be specific for a particular receptor or integral membrane protein, namely an antagonist peptide consisting essentially of at least four conservative (*sic*) amino acid residues from the amino acid sequence of at least one transmembrane domain of the integral membrane protein or a conservative amino acid substitution variant thereof", and that "[t]he specification does not teach 'random administration of random peptides' ". In contrast to Appellants' assertions, not a single claim recites what structurally definable components are to be used to effect a definable "integral membrane protein" dysfunctional in a definable "disorder", in order for the skilled artisan to reasonably know how to make and use the claimed invention (i.e., including claims 25-27 (D1 dopamine receptor), 30-31 β1-adrenergic receptor and 33-34 (α1A-adrenergic receptor)), as further argued on page 6 of the Brief. In other words, only structurally defined molecules can be "antagonists" for structurally defined integral membrane receptor proteins, by definition. Defining antagonists by "particular functional characteristics", or merely as "consisting essentially of at least four consecutive amino acid residues from the amino acid sequence of at least one [structurally unknown] transmembrane domain of the [structurally unknown] integral membrane protein...", does not address the issues concerning this aspect of the rejection; especially as it relates to claims 18, 20-24, 29, 36-37 & 60-65 in which no structurally definable peptides that are

required to practice these claimed methods are recited in any of these claims. Moreover, claims 25-26, 28, 30, 32, 33 and 35 recite "an effective analogue of fragment of (a) to (g)" which would reasonably be expected to result in inactive peptides without requiring undue experimentation to determine otherwise; consistent with the teachings of Rudinger described above, in contrast to Appellants' assertions on page 6 of the Brief. By analogy, it was held in *Ex parte Maizel* (27 USPQ2d 1662 at 1665) that:

> Appellants have not chosen to claim the DNA [product] by what it is but, rather, by what it does, i.e., encoding either a protein exhibiting certain characteristics, *or* a biologically functional equivalent thereof. Appellants' claims might be analogized to a single means claim of the type disparaged by the Court of Customs and Patent Appeals in *In re Hyatt,* 708F.2d 712, 218 USPQ 195 (Fed. Cir. 1983). The problem with the phrase "biologically functional equivalent thereof" is that it covers any conceivable means, i.e., cell or DNA, which achieves the stated biological result while the specification discloses, at most, only a specific DNA [product] segment known to the inventor. Clearly the disclosure is not commensurate in scope with the claims."

Further,

"[o]ne skilled in the art therefore cannot, as one can do with a fully described genus, visualize or recognize the identity of the members of the genus. A definition by function, as we have previously indicated, does not suffice to define the genus because it is only an indication of what the gene does, rather than what it is". *Univ. California v. Eli Lilly and Co.*, 43 USPQ2d 1398 (Fed. Cir. 1997).

Thus, in contrast to Appellants' assertions, merely reciting biologically functionally equivalent components that are otherwise required to successfully practice the method of the instant invention, does not enabled that broadly claimed under 35 USC 112, first paragraph.

Appellants argue on page 6 of the Brief that "the prior art, as described in the specification, already includes numerous examples of disorders for which administration of such antagonists is indicated. And the medical sciences will undoubtedly identify more disorders and more integral membrane proteins for which administration of such an antagonist is indicated. However, for any such indications, the present disclosure describes and enables new methods of treatment based on peptides derived from the transmembrane domains of the relevant integral membrane proteins". In contrast to Appellants' assertions, only pages 20 and 25 of the specification, and page 3 of the Brief, disclose any disorders that are associated with "over-activity" of the D2 receptors (i.e., schizophrenia), or over-expression of an EGF receptor-related oncogene (i.e., some cancers), versus any of the other alleged "numerous examples of disorders... is indicated". Further, although different art recognized adrenergic receptor antagonists may be accepted as therapeutic agents for treatment of hypertension (i.e., as described on page 23, lines 29-30), the specification provides apparently contradictory guidance on how "heart rate [can be decreased] using a β1-adrenergic-specific peptide" (page 41), because vehicle alone gave a comparable change in blood pressure when compared to administrating the β1-adrenergic-specific peptide (page 42). Therefore, treatment of hypertension as described within the instant specification does not appear to work using these adrenergic transmembrane peptide molecules (i.e., as it relates to claims 30-31 & 35); thereby, providing insufficient guidance for the skilled artisan to know how to effect any measurable phenotype for even the specific peptides of claims 30 (a-g), 31, 33 (a-g) and 34. Thus, because the specification provides contradictory evidence for

knowing how to make and use even structurally characterized adrenergic receptor peptides in the instant invention, and because no universal mechanism involving generic receptors/integral membrane proteins exists in the art for one to reasonably practice the instant invention, as claimed, undue experimentation would be required by one of ordinary skill in the art to discover how to make and use Appellants' invention, as claimed.

Moreover, an invitation for the "medical sciences" to discover what disorders are to be "indicated", and to also discover "more integral membrane proteins" to be effected, does not enable a specification under 35 USC 112, first paragraph, because it would require undue experimentation to discover such, by definition. Accordingly, enablement must be established in the specification at the time of filing and is to be commensurate in scope with the stated claims. *In re* Hogan and Banks, 194 USPQ 527 (1977). Clearly, Appellants have not conceptualized any new disorders or new integral membrane proteins that "the medical sciences will undoubtedly identify" within the instant specification. By analogy, the rejection made of record is also believed to be consistent with the court's discussion in *Genentech v. Novo Nordisk* (42 USPQ2d 1001 (Fed. Cir. (N.Y.), 1997) which held that:

"Genentech's arguments, focused almost exclusively on the level of skill in the art, ignore the essence of the enablement requirement. Patent protection is granted for an enabling disclosure of an invention, not for vague intimations of general ideas that may or may not be workable".

Finally, Appellants argue that "[o]ne of ordinary skill in the art therefore already understands the role of the transporters in a number of disorders", and that "as taught by the specification, the antagonist peptides of the invention... provide new specific therapeutic agents

useful in the transporter-related disorders as antidepressants and for the relief of drug craving and dependence". However, this restatement of pages 28-29 of the specification is the only guidance provided for the skilled artisan to know how to make and use the instant invention for these particular embodiments of the instant invention. Therefore, because it is unknown what symptoms are to be "treated" in order to "inhibit [structurally known] dopamine and/or monoamine transporters" that effect any measurable cell type, disease state, or measurable phenotype, and because administration of structurally undefined transmembrane-specific peptides are encompassed by the current claim language, the claims merely constitute an invitation to discover how to make and use Appellants' invention.

Thus, Appellants' arguments are not persuasive, for these reasons made of record.

Issue 2: Appellants argue on page 7 of the Brief that the meaning of the phrase "is indicated" ... is not indefinite and that its ubiquitous usage in medical literature demonstrates that one of ordinary skill in the art will readily understand its meaning". In contrast to Appellants' assertions, alleged "ubiquitous usage in the medical literature" does not address the rejection made of record. In other words, one reasonably cannot determine the metes and bounds of what is being "indicated" without stating such. Thus, the claims remain incomplete and ambiguous. It is also noted that Appellants do not provide arguments against the "incomplete" part of this rejection.

Appellants then argue that the phrase "consisting essentially of at least four amino acids residues" is not indefinite, especially as it relates to "conservative amino acid substitutions variants of said peptide", because "additional residues at the N- and/or C-terminus which do not alter the essential function of the peptide in the context of the invention would fall within the scope of the definition" [i.e., open claim language for any non-essential amino acid residues]. In contrast to Appellants' assertions, "scope" is an issue under 35 USC 112 first paragraph, and not an issue under 35 USC 112, second paragraph. More importantly, in that the essential feature of any peptide is its intrinsic amino acid sequence, any changes to this sequence (e.g., "conservative amino acid substitution[s]"), changes the "essential features" and peptide required to practice the invention; thereby, being indefinite and ambiguous. It should also be noted that paper no.23 (page 8) stated "claims 18 and 36 *(as well as all dependent claims)* are again indefinite" [emphasis added].

Issue 3: Appellants argue on pages 8-9 of the Brief that "[i]t is clear from a reading of the entire reference that the authors believed it was important to include not only the transmembrane domain of the relevant integral membrane protein but also portions of the specific extracellular and intracellular sequences of that protein", and that "Lofts et al. did not conceive of using peptides consisting essentially of the amino acid sequence of the transmembrane only, or of fragments of that domain, nor did they conceive of administering any portion of an integral membrane protein as a drug". In contrast to Appellants' assertions, Lofts specifically teach that

"[t]hese small proteins all include a pentapeptide from position 661-665", and that "[s]uch sequences should interact with full-length receptors and prevent receptor dimerization and thus act as specific inhibitors of function" (i.e., reasonably act as antagonists that consist essentially of at least four amino acid residues from at least one transmembrane domain, as claimed; see Abstract). In contrast to Appellants' assertions, no where in the Abstract nor discussion section of this paper is there any mention that "short extracelluar and intracellular sequences" are "required" in Lofts' method. *In arguendo,* even though portions of the extracellular and intracellular sequences of the integral membrane kinase, *neu*, were included in Lofts' peptides, these sequences are inherently not essential to practice the invention; thereby, also reasonably meeting the ambiguous claim language of "consisting essentially of at least four amino acid residues", as defined by Appellants on page 8 of the Brief for "additional residues at the N- and/or C-terminus which do not alter the essential function of the peptide". Thus, Appellants' arguments are not persuasive because Lofts teach all structural limitations for treating a disorder (i.e., tumors) in mice.

Issue 4: Appellants essentially argue on pages 9-11 of the Brief that the mechanism of action of Murphy's peptides is not the same as the instant invention, that Murphy "does not teach that peptides derived from a transmembrane domain of an integral membrane protein will act as antagonists of that protein", and that "Murphy et al. shows no data at all involving any G-protein-coupled receptor", and refers to Example 1 of Murphy (cols. 37-39). In contrast to Appellants'

assertions, columns 35-37 describe "pharmaceutical compositions" of the instant invention, as well as "treatment involves administration of the protective *composition after the appearance of the disease* [emphasis added]" (col. 35, line 65- col. 36, line 17). In particular, "inhibition of binding of Dopamine D_2 receptors" (i.e., a G-protein coupled receptor/integral membrane protein) is described, versus only ligand binding to receptors, versus that argued by Appellants. Therefore, all structural limitations of the claims are met by the teachings of Murphy, as are the functional limitations for when a "method of treating.... is indicated", in which the actions of an "antagonist" are also reasonably met by Murphy's "prevention"... "suppression"... or "treatment... after appearance of the disease" (col. 35). Additionally, in that column 36, lines 14-16, states "using a GPR polypeptide corresponding to a fragment or consensus portion of a dopamine D2 *transmembrane domain* [emphasis added]", and because column 35, lines 41-42, states that "peptides of the present invention are generally small (10-40... amino acids), the remaining structural limitations of "consisting essentially of at least four consecutive amino acid residues... from the amino acid sequence of at least one transmembrane domain" for "specifically inhibit[ing] the activity of the integral membrane protein", are inherently met. It is noted that Appellants' arguments related to single preferred embodiments of Murphy, or to their own single example of a ~~"human dopamine D2 receptor [being] disrupted by a D2-TM VII peptide" which is not~~ specifically recited in any of the pending claims, do not negate the teachings of Murphy, as it relates to the rejection made of record. Therefore, Appellants' arguments are moot, and not on point with the rejection made of record, in which no limitations are recited in the claims that

distinguishes the teachings of Murphy from the instant invention, as broadly claimed. Note that because Appellants define the limitation, "consisting essentially of at least four amino acid residues from the amino acid sequence of said at least one transmembrane domain ", as open claim language for any non-essential amino acid residues (see issue #2 above), any additional amino acid residues added to Murphy's transmembrane amino acid residues are likewise inherently not essential to practice the invention, and therefore, reasonably meet this ambiguous claim limitation of being "at least four amino acids from... at least one transmembrane domain".

For the above reasons, it is believed that the rejections should be sustained.

Respectfully submitted,

Robert C. Hayes

Robert C. Hayes, Ph.D.
November 20, 2000

Christopher S. Low
(conferee)

Michael J. Twomey
Testa, Hurwitz & Thibeault, LLP
High Street Tower
125 High Street
Boston, Massachusetts 02110

Gary L. Kunz
GARY L. KUNZ
SUPERVISORY PATENT EXAMINER
TECHNOLOGY CENTER 1600
(CONFEREE)